The Dynamics of Pulsatile Blood Flow

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The theory of dynamic fluid motion involving the forces manifested by mass, acceleration, viscous friction and vessel wall tension have been considered and studied when arterial blood flow and pressure are pulsatile. These studies and considerations have resulted in the conclusion that arterial blood pressure is dependent upon all of these parameters and that the relationship of pressure and flow is more complex and nonlinear than heretofore generally believed.

It is generally held that Poisueille’s law adequately describes the relationships of arterial blood pressure and flow. The basic assumptions underlying his law are that there is steady, laminar, parallel, flow of a Newtonian liquid in a straight, smooth walled tube. The arterial blood circulation is pulsatile, the fluid is non-Newtonian and the vessels are of complex geometry and elasticity.

Consideration of these discrepancies between idealized and actual conditions leads one to question the usual approach to hemodynamics which linearly relates flow and pressure, and which designates arterial distensibility and volume change as the primary determinants of pulse pressure.

In addition to vessel distensibility two other effects must be considered in the case of arterial blood flow which accelerates and decelerates. These are inertia and viscosity which are generally related in a complex manner to distensibility.

Considering first the effect of inertia; Newton’s second law states,

\[ F = ma \]

where \( F \) is applied force and \( m \) and \( a \) are respectively mass and acceleration. At the end of diastole the blood at the root of the aorta is essentially stationary. With the onset of systolic ejection the blood in the left ventricle and arterial tree is accelerated and may achieve a peak velocity of over 100 cm. per second within a fraction of a second. It is clear that the accelerations involved are considerable, therefore associated forces are considerable.

It is implicit from Newton’s second law that

\[ F\Delta t = m\Delta v \]

where \( \Delta t \) is an interval of time, \( \Delta v \) an interval of velocity and \( F \) is constant during the impulse. Thus, the velocity increment of a mass is directly proportional to the time interval that a constant force acts upon the mass. If the applied force is variable the impulse is obtained by integrating the force with respect to time over the involved time interval. It follows then that the instantaneous velocity due to a given applied force is inversely proportional to the mass involved.

The foregoing relationship pertains only to a frictionless system in which case the blood would be accelerated by applied cardiac force during systole, however after the heart ceased contracting the blood would continue to flow at the velocity it had achieved the instant the applied force ceased. This obviously does not occur because there is, in addition to inertia, another factor continuously affecting motion. This factor is viscous fluid friction which opposes the motion which the applied force tends to produce. It increases as velocity increases; moreover, its rate of increase is variable. Thus the acceleration resulting from an applied force is an inverse function of mass and fluid friction. Instantaneous velocity is a function of acceleration. Flow is a function of velocity.

Since arterial blood is contained within a closed system and since the inflow exceeds the outflow during systole, there is a net gain in
volume which tends to deform the vessel walls. These walls resist deformation as a function of their distensibility and thus also tend to oppose motion which applied force tends to produce. Whereas distensibility is usually given as the chief factor affecting blood pressure, it is clear that this factor is intimately related to mass and friction.

In the foregoing, the concepts of mass and acceleration, fluid friction, and distensibility have been developed separately for ease of consideration. It must be recognized that each is a variable, that each affects the other and that they are continuous functions of applied force. If these factors could be analyzed separately as they have been considered conceptually, their magnitude and behavior might be determined. If this is to be accomplished, these parameters must manifest themselves as some measurable quantity. This quantity is pressure.

Heretofore investigators have been unable to demonstrate the variability and significance of these factors within the intact arterial system.

One approach necessitates the simultaneous determination of the complex and variable time-force pattern of cardiac ejection, the mass of blood accelerated which includes the residual volume of the left ventricle, the magnitude of the accelerations, an instantaneous integrated value for all pressure drop throughout the system, and the net instantaneous distensibility of the system. This is obviously an almost impossible task.

Another approach, utilized by engineers in studying the behavior of a complex system is to drive, force, or excite motion within the system with a controlled input of known characteristics and simultaneously to measure the system’s output response. This method of analysis of the cardiovascular system appeared promising, has been tested, and is the basis of this paper. A pulse of fluid is introduced into the aorta, that is, at the source point of physiologic flow. This artificial pulse is of controlled characteristics in contrast to the unknown character of the cardiac pulse. Simultaneously the instantaneous blood pressures induced by this controlled pulse are measured in the same area. It is important to note the significance of pressure measured at the flow source. At that point the pressure drop throughout the system is integrated since it can be assumed that the pressure at the effective end is constant, with respect to time, during the controlled pulse. This simplifies the almost impossible task of measuring simultaneous pressure drop at all points in the system.

**METHODS**

A drawing of the device for causing first an acceleration followed by flow of constant velocity and then a deceleration of the intra-aortic blood is shown in figure 1.

Saline or blood at 37°C. is contained in the cylinder connected to a normally closed, rapidly acting, spring loaded, solenoid valve. A cannula connects the valve to the vessel or system to be studied. The flow from the cannula is determined by the pressure in the cylinder and the time that the valve is open. The pressures used ranged from 25 to 125 pounds per square inch.

High speed cinematography, with film speeds up to 7,000 frames per second, was used to determine the acceleration of liquid issuing from the cannula.
FIG. 2. Characteristics of fluid motion at the opening of the cannula during the time the valve of
the device, shown in figure 1, is open. (1) As valve opens flow rapidly (0.006 second) achieves a con-
stant value of 100 cc./second. (2) Since cannula orifice is constant the velocity changes from zero at
zero time and rises to 1254 cm per second in 0.006 second after which velocity remains constant until
valve closes. (3) Average velocity change divided by time for velocity change (1254/0.006) is average
acceleration in centimeters per second². (4) Product of average acceleration and volume of liquid
contained within cannula and valve assembly (mass) is equivalent to the force, in dynes, at the
orifice of the cannula.

as the valve opened, that is, the time taken to
reach constant velocity. In the experiments re-
ported this time was 0.006 second. Following this
acceleration period the issuing liquid maintained a
constant velocity, then as the valve closed, de-
celeration required 0.006 second. No evidence of
valve chatter was seen.

The mass of liquid accelerated was assumed to
be limited to the cannula and valve attachments
since they have an effective cross sectional area of
0.01 that of the cylinder. This proved to be true
since the force effects were the same whether the
cylinder was full or only 30 per cent full. The mag-
nitude of this mass was determined from the
geometry of the system and by weighing the liquid
contained therein.

Leading the output of the device, that is, chang-
ing the pressure at the cannula orifice within
limits far beyond those expected, caused no change
in the output characteristics of the pulser.

The valve can be opened and closed at will or
automatically during any part of the cardiac cycle
by means of a variable delay circuit coupled to an
electrocardiograph.*

The mass, acceleration, and volume flow within
the pulser can be varied. For most purposes the
following characteristics were used in the intra-
nortic studies: Pulse volume, 20 cc. in 0.2 second;
flow, 100 cc. per second; velocity, 1254 cm. per
second; acceleration, 2 × 10⁵ cm. per second²
(average); mass, 3 cc.; force, 6 × 10⁶ dynes. These
characteristics are plotted in figure 2.

It is important to note that this force of 6 × 10⁶ dynes is considerably more than the force it

* Circuit and valve data obtainable from author.
imparts to the blood or liquid in the vessel or chamber in which pressure is measured and to which the cannula is connected. The reasons for this difference are also important in general hemodynamic considerations. There are three such reasons.

1. The cannula orifice is 0.08 sq. cm. whereas the aortic orifice, in an average sized dog, averages 0.8 sq. cm. Since a manometer measures force per unit area the heart need to present only one-tenth the force provided by our pulser to the aorta to provide equivalent dynamic pressure changes within the aorta.

2. Having equilibrated force and pressure in the two systems of pulser to aorta and heart to aorta by a factor of approximately 10:1, we have next to deal with force transfer. A general formula has been derived\(^1\) which relates force transfer from a cylinder of one diameter to another, such as from cannula to aorta. If \(M_1\) is the effective mass of a fluid contained in a cylinder of radius \(r_1\) which is coupled to a cylinder of radius \(r_2\) and containing mass \(M_2\) then the force transfer is equivalent to the product of \(M_1\) and the fourth power of the ratio of the radii, that is,

\[
M_1 \left( \frac{r_2}{r_1} \right)^4 = M_2
\]

where \(r_1\) is the radius of the smaller cylinder. Considering the area of the aorta to be 0.8 sq. cm. and cannula 0.08 sq. cm. this is an effective loss of a hundredfold from cannula to aorta.

3. There is further loss due to friction in any system in which flow is expanding in area; such turbulence loss approaches the order of the second power.

\[
\text{Friction loss} = f \left( \text{turbulence} \left[ \frac{V_1 - V_2}{2} \right] \right)^2, \text{ character of fluid and vessel walls}
\]

\(V_1\) and \(V_2\) are the velocities in the small and large tube.

For these reasons the force of \(6 \times 10^5\) dynes within the pulser cannula induces a force within the aorta of far less than this value for there is great loss as compared with loss from heart to aorta. Since the effective orifice from heart to aorta is essentially 1:1, the losses are very much smaller. Thus, the pressure within the ventricle and aorta during systole are similar, whereas the pressure within the pulser is very much greater than in the aorta during the artificial pulse.

Capacitance manometers coupled with catheters were used to record pressures. The dynamic responses of the pressure recording systems were tested frequently and were over 100 cycles per second. In these experiments the catheter was maintained just distal to the injection cannula. The bulb on the end of the cannula and the orientation of the cannula orifice insured dissipation of jet effects to the extent that these did not cause spurious pressure changes.

**Comparison of Theory and Experimental Findings**

**Theory**

Forces are rigorous physical terms and vector quantities, however in this paper the pressure manifestations of the effects of mass accelerated, friction and distensibility will be termed
If the described artificial pulse were introduced into a tube containing liquid but whose walls were not increasingly resisting deformation, that is, were not distended at any time during the pulse, and if there were no viscous friction, the only force existing within the tube during the pulse would be $F_{(ma)}$. Even though liquid were running into the tube during the entire pulse all that would be seen on the recorded pressure would be the pattern of $F_{(ma)}$ as shown in the uppermost curves of figure 3.

If, next, the pulse were introduced into a system wherein viscous friction developed, such frictional force would increase at some rate during acceleration, remain constant when velocity became constant, and decrease during deceleration. If this tube were still undistended the pressure pulse pattern would be determined by the instantaneous combination of $F_{(ma)}$ and $F_{(d)}$ as shown in the middle curves of figure 3.

If the pulse were introduced when the tube walls were actively stretched or distended, we should find a third force, $F_{(k)}$, in addition to the previous two. As the volume of the tube becomes greater during the pulse and the walls are stretched more and more, $F_{(k)}$ also increases. The recorded pressure pulse would then be again the instantaneous combination of these three forces as seen in the lower curves of figure 3. The initial rise in pressure which is caused by the acceleration of mass and developing viscous friction will henceforth be termed the acceleration transient.

There are three basic inaccuracies in the highly schematic drawings shown in figure 3:

1. Development of forces are shown as linear plots whereas they are actually curves;
2. Acceleration and deceleration plots, as drawn, constitute a considerable period in the total pulse length; actually they are very short compared to the total length of the pulse;
3. Volume flow actually begins at the instant acceleration starts; thus, $F_{(d)}$ has some finite value at the end of acceleration. To demonstrate the time course of these events more clearly a hypothetical force and pressure diagram is shown in figure 4.

### Experimental Results

1. **Schema Experiments.** A mechanical model was constructed in which the pulser could be

![Fig. 4. Hypothetic force and pressure curve to demonstrate, more closely, the actual changes occurring in $F_{(ma)}$, $F_{(d)}$, and $F_{(k)}$ during acceleration. Since the total pulse length is long, compared with acceleration, the final value of pulse pressure at the end of the pulse depends upon the length of the pulse, rate of flow, viscous friction and distensibility. The course of pressure at the end of pulse (broken line above c) depends upon the pressure level achieved at the end of the pulse and thence upon the reverse force effects, that is, deceleration, decrease in viscous friction and flow. (A), time required for acceleration. (B), time duration of entire pulse following (A). (A) is very short compared with (B) [(A) 0.006 second, and (B) 0.2 second]. $F_{(ma)}$, $F_{(d)}$, and $F_{(k)}$ vary with respect to each other.](image-url)
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FIG. 5. Lower curve, actual pressure pulse recorded by capacitance manometer from rubber tube through which water was flowing. Positive pressure record, cross hatched for easier visualization. Upper curve, hypothetic plot of forces producing the actual pressure pulse. Discussion in text.

Fig. 6. Copy of recorded pressure pulse obtained from the same rubber tube from which the record in figure 5 was taken. Tube became distended at F(k).

coupled into tubes of varying distensibility through which saline or blood would flow under various conditions. In this way certain factors could be held constant while others were varied. Figures 5 and 6 are actual pressure records which have been selected because they demonstrate, simply, the actual effects predicted from theory. When the injected pulse was introduced into an undistended rubber tube through which blood was flowing, the induced pressure pulse was as shown in the lower curve of figure 5. It can be seen that, primarily, two forces are involved: There is first an acceleration transient which is the sum of \( F(ma) \) and \( F(V) \). After acceleration is complete, and flow remains constant, the pressure remains elevated due to the constant opposing force of viscous friction. Superimposed upon this sustained pressure elevation are reflected waves.

The tube is not distended; thus, even though flow into the tube is continuous, there is not a continuous rise in pressure. Here, then, is a pressure pulse with no element of increasing wall tension or "volume-distensibility." The upper curve of figure 5 is a graphic drawing to demonstrate the force-time distribution which produced the pressure pulse.

Figure 6 was obtained from the same tube as it became distended during the pulse. In this case the flow from the pulser was greater, thus both the acceleration transient and viscous friction were greater. At the arrow labeled \( F(k) \), the tube became distended, and since flow continued, the pressure continued to rise as the tension within the walls of the tube increased.

In this case all three forces were in evidence. \( F(k) \) is added to, that is, superimposed upon, the persisting \( F(V) \).

Such schema studies revealed that these forces are actually additive, thus when a second pulsing cannula was added to any system and the two pulsed simultaneously the resultant pressure wave was the sum of those obtained separately. If the characteristics of the model were changed in relationship to the geometry or elastic quality of the tubes, the magnitude of these force effects, for the same pulse, were likewise altered. These alterations were seen, not only in \( F(k) \), but also in the other two forces.

When very distensible tubes were used in the model the effective mass and viscous friction were so reduced that no induced pressure pulse was seen. The corollary is also true, that in rigid tubes these forces become very great.

2. Experiments on the Intact Vena Cava. When artificial pulses are applied to the intact abdominal vena cava of living dogs certain interesting characteristics of the venous circulation are revealed which further substantiate the theoretic concepts of force and pressure.

Figure 7 shows a series of typical pressure curves obtained from an experiment upon an anesthetized dog. Curve A was obtained under "normal" venous circulatory circumstances. There is initially an acceleration transient followed by a low sustained pressure, then a deceleration transient. In this case the main force
is $F_{(ma)}$, viscous friction is very low and the vein walls are not distended.

Curve $B$ is a pulse obtained midway between the renal and femoral veins after the vena cava had been constricted below the renal veins, thus impeding outflow distal to the point of occlusion. The acceleration transient has approximately the same magnitude as in curve $A$ and the sustained elevation during the period of constant velocity is greater. Further, the vein was still not distended.

Curve $C$ was obtained after further occlusion had caused venous distention. In this case all three forces are again in evidence. First, there is the acceleration transient and the sustained viscous friction force upon which wall tension is superimposed.

3. Experiments upon the Intact Aorta. Dogs, anesthetized with a mixture of Nembutal, Dial and urethane, were used. Their weights varied from 5 to 20 Kg. Their apparent age varied from puppies to old dogs. The pulse cannula connecting the solenoid valve with the ascending aorta was inserted through a carotid artery (A) (B) (C)

**Fig. 7.** Actual pressure curves from the intact abdominal vena cava of a dog. (A) When the vein and its internal blood flow was "normal." (B) After partial occlusion but before distention. (C) After vein wall had become round and distended.

Saline pulses were then injected into the aorta at various phases of the cardiac cycle. Thus, the pressures in the aorta resulting when fluid was accelerating, moving at constant velocity or decelerating could be studied, for there was only a single period of the pulse when these respective conditions existed. The input pulse and the animal's vascular system could be treated as two separable variables, that is, the pulse characteristics could be kept constant and the animal's cardiovascular status varied or vice versa.

Figure 8 shows the characteristics of the pressure pulses due to cardiac action and likewise those induced by the pulsar's action. The following characteristics of the induced pulse should be noted. Initially the pressure rises abruptly. This could not be due to the relationship of increasing volume and vessel wall stretch, since essentially no liquid has entered the aorta in this short period. The rise is due to the two forces, $F_{(ma)} + F_{(f)}$, indicated previously. Following the acceleration transient, the pressure continues to rise from that level at low in the neck. Catheters for transmitting pressure changes were inserted into the carotid or femoral arteries and moved under fluoroscopic guidance until one catheter came to lie in the ascending aorta adjacent to the pulse cannula, and the second catheter in the distal end of the aorta at the level of its bifurcation. These positions were later checked at the time of the postmortem examination; foreshortening of the arterial system occurs when intra-arterial pressure falls.

**Fig. 8.** Upper record, pressure pulse from the ascending aorta of a dog. Lower record, simultaneous pressure pulse 25 cm. lower in the pelvic aorta.

I, onset of the systolic rise in pressure due to cardiac activity. II, onset of artificial pulse, that is, acceleration transient. III, rise in pressure due to $F_{(a)}$, superimposed on $F_{(f)}$. The slope of this rise is due to net volume gain and simultaneous distensibility. IV, onset of deceleration and the end of the induced pulse. V, onset of next cardiac systole. See text for description of other symbols.
a markedly slower rate. This increase in intra-aortic pressure is superimposed upon the persisting $P(t)$ and is due to $F(v)$. It is proportional to the net gain in volume within the system and the vessel's effective distensibility. Note the similarity of the induced and cardiac pressure pulses.

Calculation of these parameters are thus possible in the following manner:

$$\text{(A)} = \text{Rate of pressure rise due to injected volume minus simultaneous run-off} = \frac{\Delta P_0}{\Delta t_0}. \text{This is the slope of the curve during the period of constant velocity injection.}$$

$$\text{(B)} = \text{Rate of pressure fall in similar pressure ranges due to run-off} = \frac{\Delta P_0}{\Delta t_0}. $$

**Fig. 9.** Four sets of double channel recordings of arterial blood pressure. Upper record in each pair was obtained from the ascending aorta while the lower record was simultaneously recorded from the femoral artery. Vertical lines are 5 mm. apart. Paper speed was 125 mm. per second in all records. Pressure in millimeters Hg is noted on the left margin of each aortic pressure record. Discussion in text.
\[
(C) = \text{Rate of increase in volume injected with respect to time } = \frac{\Delta V}{\Delta t}.
\]

\[
\frac{(A) + (B)}{C} = \text{pressure per volume ratio, since at each instant of time the sum of } A + B \text{ is the pressure change which would have resulted from an equal incremental change in volume corrected for run-off. This is the reciprocal of distensibility.}
\]

\[
\frac{(B)}{\Delta P/\Delta V} = \text{Rate of run-off during this period.}
\]

Thus, the magnitudes of the acceleration transient, sustained viscous friction, and effective distensibility of the arterial system at the site of injection can be determined and, in addition certain characteristics of the pressure pulse due to cardiac activity and run-off can be deduced.

Figure 9 shows simultaneous aortic and femoral pressure pulses from one experiment. There are four sets of two channel records. The uppermost pair were obtained during a period when the animal’s blood pressure was “normal”; no drugs other than the anesthetic agents had been given. The top record shows three successive intra-aortic pressure pulses of cardiac origin, next the induced pressure pulse (note signal marker on bottom of record) followed by two additional pulses of cardiac origin. The lower pulses in the upper record were obtained simultaneously from the femoral artery.

The second series of aortic and femoral pressure tracings were recorded from the same animal following a rise in blood pressure caused by the administration of epinephrine. The third series was obtained following the administration of acetylcholine which caused a marked fall in blood pressure. The bottom series were recorded during severe hemorrhagic hypotension. The only pressure pulses seen in the lower records are those due to the artificial pulse.

Figure 10 shows five accurately traced records from another animal. They are presented because the germain characteristics of the curves are more easily seen. Curve B was obtained under “normal” conditions, curve A following epinephrine administration, curve C during acetylcholine-induced hypotension, curves D and E at lower blood pressure levels following bleeding.

As blood pressure rose above the control level, an injected pulse having the same characteristcs as before induced a pressure pulse which has the following differences as compared with those seen under normal circulatory conditions: (1) The acceleration transient increases in magnitude as the blood pressure rises even though the input force from the pulser remains constant. (2) The pressure-volume slope increases in relation to the decreased rate of run-off and also to the altered distensibility. The relative proportions of these effects vary with the drug and its dosage. (3) The deceleration transient is likewise increased in magnitude and is again related to the change in blood pressure.
As blood pressure fell below control levels, the acceleration transient increased in magnitude above the control level as the blood pressure fell. In the case of hypotension due to acetylcholine there was marked vasodilation and very rapid run-off to such a degree that there was no rise in pressure during the pressure-volume phase, that is, fluid was running out of the segment of vessel as rapidly as it was running in, yet the acceleration transient bore a close relationship in magnitude to that seen at an equal blood pressure level during hemorrhagic hypotension, in which case there is marked vasoconstriction, as seen in the steepened slope of the pressure-volume curve.

Figure 11 is a composite curve obtained from a group of 15 middle age dogs with weights ranging from 14 to 18 Kg. The characteristics of this curve which are noteworthy to this discussion are: (1) As the product of mass and acceleration of the injected pulse is increased, for any given diastolic pressure, the acceleration transient is likewise increased but in a nonlinear manner. (2) As the blood pressure ranges from low to high levels, the acceleration transient varies in a very interesting U-shaped curve. The trough of this curve tends to fall at a level of approximately 90 mm. Hg, if the acceleration value for the injected pulse remains with a value of approximately $10^4$ cm. per second$^2$. In large or old dogs the minimum of the trough tends to move to the right, that is, to higher pressure levels. If the acceleration value is increased the trough of the curve also moves to the right, but if acceleration is reduced its minimum moves to the left. Likewise, if the mass which is accelerated, at any given rate, is increased, the trough moves to the right, and vice versa.

The relationships of the pulser cannula to the recording catheter have been studied. If the orifice of the latter faces that of the cannula, artefacts appear due to vibration of the catheter in the ejected stream. The same artefact arises when the recording catheter is placed facing the aortic valve orifice. When it is moved distally, the induced wave changes its shape just as the natural pulse does. This means that the recorded pressure pulse is not distorted by the issuing stream.

**DISCUSSION**

Data obtained from mechanical models, intact veins and arteries indicate that the pressure pulses, induced by applied input flows of similar pattern, vary considerably in their character and magnitude. It is evident that the relationships of flow and pressure are likewise variable. The interacting parameters responsible for this variability have been broken down into the effects of mass, friction and distensibility.

From the curve of lumped effects shown in figure 11 it is evident that the relationship of flow and pressure in the arterial system is decidedly nonlinear when the artificial pulse is injected into the aorta. It appears that effective mass and friction increase markedly on either side of the null point to produce this effect. Whether the pressure effects of these parameters are additive in a precise algebraic manner in the circulatory system as they appear to be in the model, and as have been indicated graphically, is speculative. It is certain, however, that the initial rise and sustained pressure designated as mass and friction are just that, and the variability of their cumulative total indicates their importance. It has been shown that the effective force applied to the intra-aortic blood column by the pulser is very much less than that applied by the left ventricle under normal conditions. It seems valid, then, to assume that blood flow of cardiac origin is also nonlinearly related to pulse pressure. Since the heart may possess the property of modifying its force pattern relative to the
characteristics of the recipient vascular system the next step is to determine the magnitude of the nonlinear relationship of physiologic cardiac output and arterial blood pressure.

The initial systolic rise of intra-aortic pressure is sudden and of significant magnitude. This has been difficult to explain for, at best, only a very small volume of blood could be expelled from the heart into the aorta during this brief period. The static distensibility of the vessels is such that this small volume could hardly have caused such a pressure rise, per se. Hamilton and his associates offered an explanation of this phenomenon which seemed the best at the time: that is, the aorta could not stretch rapidly enough to take up even this small volume. While their observations on the "reluctance to rapid stretch" of the aorta may be true, a more complete explanation for the initial pressure rise can be offered. With the onset of mechanical systole, acceleration of blood must occur. It has been shown that acceleration of liquid within rubber tubes, veins and arteries manifests a pressure rise which is only indirectly related to volume change and distensibility. Indeed this initial rise in pressure occurs in vessels whose walls are not stretched by increasing internal volume. Thus, it can be predicted that such a rapid initial rise in pressure occurs as a result of the systolic acceleration of blood.

There are not, to the author's knowledge, data in the literature accurate enough to demonstrate the magnitude of the mass of blood accelerated or the acceleration provided by the heart. The residual ventricular volume and arterial volume have not been measured during life. Stroke volume is not measured accurately enough. According to certain data the static arterial blood volume in a 14 Kg. dog is 60 cc. at 80 mm. Hg, 25 cc. at 40 mm. Hg, and 106 cc. at 150 mm. Hg. Such figures are indicative of the large changes in arterial volumes relative to pressure. Even if these values were known, they do not constitute the effective mass which is also a function of distensibility. Acceleration itself has been, by different approaches, variously estimated to be from $1 \times 10^3$ to $2 \times 10^4$ cm. per second. Many of these values have been obtained from vessels other than the ascending aorta. Turbulence further modifies the relationships. These are problems in which errors in milliseconds, milliliters and millimeters are significant. Effective distensibility during life and under various conditions have likewise been difficult to measure. Conclusions are based on values obtained from dead animals and man after the small vessels have been ligated. Diodrast has been injected into the aorta of living animals and estimates of volume have been obtained from radiographic pictures and correlated with pressure. We have found that effective distensibility varies considerably during life and that postmortem distensibility depends upon how the animal was killed. Pulse wave velocity is apparently not a sole function of distensibility but probably depends also upon pulse amplitude.

A different approach to understanding the interplay of cardiovascular forces lies in the comparison of the pressure pulses induced by our artificial pulser, and by the heart under similar circumstances, as shown in figures 9 and 10. Similarities in contour between the natural and artificially induced pulse are apparent. It is reasonable to assume that similar forces are involved in both pulses. It is their relative magnitude and time relationships which are in question.

Direct Fick determinations were made during most of these studies at the time the artificial pulses were induced within the aorta. If such determinations were accurate, if all pulses of cardiac origin were identical in length and stroke volume, and if the recipient arterial system did not change during the Fick determination period, it would be a simple matter to obtain a derived figure for stroke volume. The effective distensibility could be calculated by the method outlined earlier in this paper and a correction could be made for run-off during systole. The value for the net volume increase during systole could then be substituted into the ratio of calculated distensibility and the expected increase in systolic pressure could be derived. This value could then be compared with the actual pressure rise obtained. The difference would be due to $F(ma)$ and $F(D)$. In most
instances few, if any, of these conditions could be satisfactorily met. There were, however, certain occasions where oxygen consumption was constant, where both arterial and venous oxygen content was unchanged in duplicate determinations, and at the same time pulse rate and pulse pressure contour did not noticeably vary. Records obtained under these conditions have been analyzed in the manner described. The measured pulse pressure was, in all cases, from 15 to 20 per cent higher than the pressure predicted from calculated net volume change and distensibility. The pulse pressure induced by the artificial pulser was also greater and the difference corresponded to the magnitude of the acceleration transient. Furthermore records in figures 8 and 9 obtained under conditions of hypo- and hypertension suggest that the magnitude of \( F_{ma} \) and \( F_U \) should likewise be increased relative to stroke volume. For example, note that in figure 10c, the induced pressure pulse has a sharp initial rise with a peak which falls back and then continues at a more or less constant level. This would indicate that fluid was flowing out of the aorta as rapidly as it was flowing in. The entire pressure pulse then is due only to \( F_{ma} \) and \( F_U \) and there is no evidence of \( F_{d} \). Note too that the pulses of cardiac origin have a rapid initial rising pressure a sharp peaking break and no further rise. The pressure then falls rapidly to the incisura as if it were "collapsing" after the applied force ceased; apparently it was being sustained by \( F_U \) which is set by \( F_{ma} \). In the case of both natural and artificial pulsations there is a stroke volume producing the pulse pressure; however, in neither is there evidence of vessel distention. The measurement of cardiac output by the Fick method, under these circulatory conditions, was unsatisfactory hence a check on these assumptions by this method, was impossible.

The difficulty of calculating stroke volume from pressure pulse prove to be reasonably accurate under experimental conditions, the heart must, in some unknown way, modify its force pattern so that the total sum of these nonlinear forces are kept constant and hence do not introduce a recognizable and variable error. Many workers believe that the reliability of the Fick principle is probably not better than within 15 or 20 per cent under ideal conditions. We must, therefore, await a method for determining arterial blood flow which is reliable under all of the cardiovascular conditions which we wish to investigate.

It is impossible to include all of the data obtained from these experiments in a single paper. However, one additional interesting facet of this study can well be included. In figure 11 the minimum point of the curve is at 90 mm. Hg. The ordinate values of this curve represent, we believe, the forces \( F_{ma} \) and \( F_U \). Since mass and friction oppose changes in motion for any given applied force, it appears that the values of these effective forces are minimal at that pressure level. In any mechanical or electrical circuit there is a null operational level at which there is maximum power transfer. The circulatory system should automatically seek this level of optimal operation unless driven by other forces. It is reasonable to propose this as an explanation for normal blood pressure being what it is and not some other value. Interestingly enough, Hamilton and Remington found in their studies on dead dogs that the maximum distensibility occurred between the levels of 80 and 100 mm. Hg. It has been shown, herein, that increasing distensibility decreases effective mass and viscous friction.

This paper has dealt with the qualitative aspects of these effects. The difficult task of rigorously evaluating their quantitative characteristics is now in progress. Hydrodynamacists have found it difficult to write differential equations describing the behavior of these parameters in systems of defined character. If such equations could be written for the complex cardiovascular system hemodynamics would be definable in rigorous terms and our approach to circulatory physiology would be greatly facilitated.
SUMMARY

1. The theory of dynamic fluid motion predicts that in the arterial system, where pulsatile flow occurs, three parameters are involved: (a) the pressure manifested by the product of mass and acceleration \( F_{ma} \); (b) that manifested by viscous friction \( F_f \); (c) that manifested by wall or spring tension \( F_s \), 1/dis-tensibility.

2. Since pressure is force per unit area, arterial pulse pressure should be the instantaneous sum of these forces. Since some of these parameters are functions of flow while others impede flow, the relationship of pressure and flow becomes complex due to mass and friction. If these parameters vary with respect to each other in a nonlinear manner, this relationship is, with present knowledge, unpredictable.

3. Since the heart develops a variable and unknown force pattern, an artificial pulser was built which would supply the arterial system and its contents with a known and constant force pattern. In this way the characteristics of the arterial system and their influence on effective arterial mass, viscous friction and distensibility could be studied.

4. Data are presented which show: (1) that these three parameters vary in a nonlinear manner and each affects the other; (2) that vascular pulse pressure is the sum of these forces; (3) that pulse pressure varies considerably in magnitude and in its component forces under various circulatory states even though "stroke volume" remains constant.

5. The reasons why these significant factors have escaped identification are discussed.

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